Complete Summary

GUIDELINE TITLE

Myocardial infarction.

BIBLIOGRAPHIC SOURCE(S)

Finnish Medical Society Duodecim. Myocardial infarction. In: EBM Guidelines. Evidence-Based Medicine [CD-ROM]. Helsinki, Finland: Duodecim Medical Publications Ltd.; 2003 Jul 11 [Various]. [30 references]

COMPLETE SUMMARY CONTENT

SCOPE

DISEASE/CONDITION(S)

- Myocardial infarction
- Arrhythmias associated with myocardial infarction, including ventricular fibrillation, ventricular tachycardia, ventricular ectopic beats, idioventricular rhythm, supraventricular tachyarrhythmias, bradyarrhythmias
- Circulatory conditions associated with myocardial infarction, such as hyperdynamic state, neurovascular reflex (bradycardia-hypotension), hypovolemia, and severe heart failure

GUIDELINE CATEGORY

Diagnosis
Evaluation
Management
Risk Assessment
Treatment

CLINICAL SPECIALTY

Cardiology
Family Practice
Internal Medicine

INTENDED USERS

Health Care Providers
Physicians

GUIDELINE OBJECTIVE(S)

Evidence-Based Medicine Guidelines collect, summarize, and update the core clinical knowledge essential in general practice. The guidelines also describe the scientific evidence underlying the given recommendations.

TARGET POPULATION

Individuals with suspected or definite myocardial infarction

INTERVENTIONS AND PRACTICES CONSIDERED

Diagnosis/Evaluation

1. Evaluation of signs and symptoms (e.g., pain)
2. Electrocardiograph monitoring
3. Measurement of myocardial enzymes (creatine kinase [CK], creatine kinase-MB [CK-MB], creatine kinase-MB mass [CK-MB mass], cardiac troponins T and I)
4. Blood hemoglobin, leukocytes, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP)
5. Serum sodium and potassium and chest x-ray, if needed

Treatment of Myocardial Infarction

1. Oxygen
2. Glyceryl nitrate (mouth spray or sublingual tablet), intravenous morphine or oxycodone
3. Beta-blockers, such as metoprolol, atenolol, practolol
4. Acetylsalicylic acid (aspirin)
5. Thrombolytic therapy, such as streptokinase or accelerated tissue plasminogen activator
6. Angiotensin-converting enzyme inhibitors, such as captopril
7. Percutaneous transluminal angioplasty (with or without stent insertion)
8. Continuous nitrate therapy, such as isosorbide dinitrate
9. Heparin
10. Warfarin

Treatment of Infarction-related Arrhythmias

1. Cardiac monitoring
2. Defibrillation or cardiopulmonary resuscitation followed by defibrillation

3. Lidocaine
4. Amiodarone
5. Beta-blockers
6. Digitalis
7. Direct current shock
8. Pacemaker
9. Atropine

Note: Guideline developers considered, but did not offer recommendations for, ibutilide.

Treatment of Circulatory Conditions

1. Saline
2. Beta-receptor blockers, such as metoprolol, atenolol, or practolol
3. Atropine and dopamine infusion
4. Nitrates
5. Continuous positive airway pressure
6. Treatment of pulmonary oedema

Assessment of Risk Factors

1. Evaluation of risk factors for mortality
2. Evaluation of ischaemia and need for coronary surgery or angioplasty

Follow-up Care

1. Drug therapy including aspirin, beta-blockers, angiotensin-converting enzyme inhibitors, diuretics, nitrates, and anticoagulants
2. Statins and cholesterol-lowering diet
3. Folic acid (both vitamin B6 and B12 to lower serum homocysteine concentrations)
4. Measurement of serum lipids
5. Counseling for a healthy lifestyle
6. Evaluation of symptoms and ability to return to work

MAJOR OUTCOMES CONSIDERED

- Short-term mortality
- Rate of reinfarction
- Recurrent ischaemia
- Cardiac death
- Vascular death
- Mortality
- Sudden cardiovascular death
- Frequency of strokes
- Incidence of adverse effects, including major bleeding
- Accurate diagnosis of myocardial infarction
- Sensitivity and specificity of diagnostic/prognostic tests

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Hand-searches of Published Literature (Primary Sources)
Hand-searches of Published Literature (Secondary Sources)
Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

The evidence reviewed was collected from the Cochrane database of systematic reviews and the Database of Abstracts of Reviews of Effectiveness (DARE). In addition, the Cochrane Library and medical journals were searched specifically for original publications.

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Weighting According to a Rating Scheme (Scheme Given)

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Levels of Evidence

A: Strong research-based evidence. Multiple relevant, high-quality scientific studies with homogeneous results.

B: Moderate research-based evidence. At least one relevant, high-quality study or multiple adequate studies.

C: Limited research-based evidence. At least one adequate scientific study.

D: No research-based evidence. Expert panel evaluation of other information.

METHODS USED TO ANALYZE THE EVIDENCE

Systematic Review

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not applicable

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Not applicable

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

Not stated

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

The levels of evidence [A-D] supporting the recommendations are defined at the end of the "Major Recommendations" field.

Objectives

- If a person at risk of a myocardial infarction (MI) has an acute coronary syndrome lasting over 20 minutes, an imminent MI must be suspected. Instead of chest pain, acute dyspnoea may be the primary symptom.
- An acute coronary syndrome without myocardial damage is often unstable angina, which calls for active treatment.
- The diagnosis should be made without delay since early therapy improves the prognosis decisively.
- Thrombolytic therapy is given as early as possible in all cases with a clinical picture of imminent MI and corresponding electrocardiogram (ECG) changes. See the Finnish Medical Society Duodecim guideline "Thrombolytic Therapy in Acute Myocardial Infarction."
- Acute angioplasty (percutaneous transluminal coronary angioplasty [PTCA], percutaneous coronary intervention [PCI]) is an alternative or a complementary procedure to thrombolytic therapy ("Primary angioplasty versus intravenous..." 2002; Sim, et al., 1995; DARE-953385, 1999; Grines, et al., 1999) [A]. Angioplasty is probably preferred, at least in ST elevation MI (Keeley, Boura, & Grines, 2003).
- If there are no contraindications, acetylsalicylic acid (ASA, aspirin) and a beta-blocker should be started for all patients and, for most patients, also an angiotensin-converting enzyme (ACE) inhibitor and a statin on the first days of treatment.
- Health care system should include a planned care pathway for coronary patients.

Diagnosis

- The diagnostic criteria change in the course of treatment.
    - During first aid, pain is the primary symptom in younger patients. Presentation in the elderly is often atypical.

        - When thrombolytic therapy is considered, an ST elevation on the ECG or a recent left bundle branch block (LBBB) should be taken into account. See the Finnish Medical Society Duodecim guideline "Thrombolytic Therapy in Acute Myocardial Infarction."
        - In addition to pain and ECG findings, myocardial enzyme levels are needed for definite clinical diagnosis.
- For differential diagnosis of chest pain, see the National Guideline Clearinghouse (NGC) summary of the Finnish Medical Society Duodecim guideline Differential Diagnosis of Chest Pain.
- The pain in MI lasts over 20 minutes and is localized widely in the retrosternal area, with radiation to the arms, back, neck or lower jaw. The pain is squeezing and is experienced as tightness, heaviness, and pressure or pressing. Breathing or changing posture does not influence the intensity of pain. The pain is usually severe and consistent. It may be localized in the upper abdomen, in which case, if nausea and vomiting are also present, it simulates acute abdominal disease. The patient is often pale, in a cold sweat, and anxious.
- MI may also present as acute pulmonary oedema, spells of unconsciousness, or sudden death.
- Thrombolytic therapy is indicated
    - if the pain has lasted less than 6 to 12 (24) hours and there is at least a 2-mm elevation in the ST segment in at least two chest leads, or
    - a 1-mm elevation of ST in at least two leads in the extremities, or
    - a reciprocal ST depression in V1-V4, or
    - a recent left bundle branch block
- The contraindications for thrombolytic therapy must always be considered. See the Finnish Medical Society Duodecim guideline "Thrombolytic Therapy in Acute Myocardial Infarction."
- In clinical investigation, remember that the ECG and myocardial markers change with the course of the disease: first there is an ST elevation, after that development of the Q-wave, and finally T-wave inversion. Complications must also be recognized. In a T-wave infarction (non-Q-wave infarction), no classical Q waves are present, but the diagnosis is based on an increase of myocardial enzymes, chest pain, or ST-T changes. Classical Q-wave changes, ST elevations, and T inversions may be caused by various other diseases, which should be remembered in the differential diagnosis. An old infarction, bundle branch block, and early repolarization make the diagnosis difficult, in which case the change in ECG is important and an old ECG recording valuable. When added to other criteria, "minor" signs of infarction are also important.
- The European Society of Cardiology and the American College of Cardiology have agreed on a new definition of MI (Yusuf et al., 2000):
    - Typical increase in the concentration of serum cardiac troponins or creatine kinase isoenzyme containing M and B subunits (CK-MB) associated with at least one of the following:
        - symptoms of cardiac ischaemia
        - recent pathological Q waves in the ECG
        - ischaemic ST segment changes in the ECG
        - coronary artery revascularization

ECG Diagnosis

- Points for taking an ECG: acute care, emergency room, 12 hours later, on day 2, upon discharge from hospital and thereafter as deemed necessary.
- ECG is the most important diagnostic procedure. To start with, the positions of the chest leads must be marked on the skin to allow detection of meaningful changes on the ECG. By monitoring the ECG, the efficacy of the treatment can be assessed. However, in the early stages there may be no changes in ECG, and the changes may be first evident after hours or even days. An ECG diagnosis is made more difficult by an old infarction, left bundle branch block, or posterior infarction.
- In posterior wall infarction, a reciprocal ST segment depression in V1-V4 simulates ischaemia. A posterior infarction is, however, often inferoposterior and, in addition to ST segment depression, ST segment elevations are found in leads III and aVF.
- ST depression is suggestive of ischaemia and/or unstable angina pectoris. Extensive ST depressions in connection with a clinical picture of MI can indicate subendocardial damage.

Tests Following the ECG

- Troponin is the most important new marker and is replacing creatine kinase (CK).
- CK and CK-MB or CK-MB mass (CK-MBm).
- A negative troponin T, troponin I, or CK-MBm result 9 to 12 hours after the onset of symptoms practically rules out MI.
- Troponin T test is also valuable, if the time lapse since the beginning of the symptoms is more than 24 hours (the concentration remains elevated longer than that of CK). An elevated troponin T or troponin I concentration predicts adverse events irrespective of ECG findings (Olatidoye, et al., 1998; DARE-981100, 2000) [A].
- The tests should be performed 3 times in case of suspected infarction: on arrival of the patient and 12 and 24 hours after arrival.
- Blood haemoglobin, leukocytes, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP)
- Serum sodium and potassium, and chest radiograph if needed

Troponin-T or Troponin-I

- Principal indicators of myocardial damage, which can also be determined by means of rapid testing methods suited for primary health care. A reading device facilitates the interpretation.
- Troponin is more myocardium-specific than CK-MB and is also very sensitive.
- The concentration increases rapidly (in 4 to 6 hours) after myocardial damage, and the elevated levels persist for at least one week.
- Indications:
    - To verify or exclude MI (or myocarditis) when at least 6 hours have elapsed from the onset of pain. Unstable angina pectoris may give positive results, indicating slight myocardial damage, which means that the prognosis is serious regardless of the ECG findings and active treatment is necessary. The normal reference concentration is zero, or the method-dependent threshold is often given as
    - A negative result within 12 hours after the onset of pain excludes infarction.

    - Also used for the diagnosis of infarction when the patient's arrival for treatment is delayed, and CK and aspartate aminotransferase (AST) have returned to normal.
    - Troponin verifies MI in cases where high CK concentration from skeletal muscle increases the CK-MB concentration over normal limits.
- Mild elevations in the concentration that exceed the threshold are often seen in cardiac surgery. The threshold level that would verify the diagnosis of MI has not been defined for the situations. Mild elevations may also occur in connection when prolonged tachycardia causes a strain on the sick heart.

Serum CK-MB Mass

- More specific and sensitive than CK-MB
- Abnormal within 6 to 8 hours from the beginning of the pain, and remains abnormal for 1 to 2 days.
- Slightly positive values may indicate mild myocardial damage that requires active treatment. Unlike with troponin, the normal concentration of CK-MB is not zero. There is an uncertain borderline area of 5 to 10 micrograms/L between the positive and negative result.

Myoglobin

- Reacts most rapidly to myocardial damage and is positive from the first hours onward
- Not a specific indicator of myocardial damage. Negative myoglobin is valuable in exclusion diagnosis.
- Lack of reference values limits use.

Differential Diagnosis

- The most important differential diagnoses include
    - myopericarditis
    - aortic dissection
    - pulmonary embolism
    - unstable angina pectoris
    - oesophageal pain
- See the NGC summary of the Finnish Medical Society Duodecim guideline Differential Diagnosis of Chest Pain.

Treatment

- Oxygen, if there are problems in oxygenation (pulmonary oedema).
- For treating pain
    - Glyceryl nitrate: mouth spray or sublingual tablet
    - Morphine 4 to 6 mg intravenously (i.v.), additionally 4 mg 1 to 3 times at 5 min intervals, if necessary. Oxycodone 3 to 5 mg i.v. is an alternative.
    - A beta-blocker (metoprolol, atenolol, practolol) 2 to 5 mg i.v. may sometimes ease the pain.
- ASA 250 mg, chewable tablet or dissolved in water, unless there are contraindications (active ulcer, hypersensitivity to ASA, anticoagulation)

("Collaborative overview of randomised trials of antiplatelet therapy" 1994; DARE-948032, 1999) [A].

- A beta-blocker (Danchin, De Benedetti, & Urban, 2002) [A] is always instituted, unless there are contraindications (asthma, hypotension, heart insufficiency, conduction disturbance, bradycardia). The first dose can be given intravenously if the patient is in pain, or orally if the patient is pain-free and time has passed since the infarction. Beta-blockers are useful especially in patients who are tachycardic and hypertensive but do not have heart failure.
    - i.v. dose: metoprolol or atenolol 5 mg
    - Orally, metoprolol or atenolol 25 to 50 mg x 2
- Thrombolytic therapy, unless there are contraindications ("Indications for fibrinolytic therapy...", 1994; DARE-948029, 1999) [A].
- Immediate percutaneous transluminal angioplasty ("Primary angioplasty versus intravenous...", 2002; Sim, et al., 1995; DARE-953385, 1999; Grines, et al., 1999) [A] may be performed when thrombolytic therapy is contraindicated. The effect is better than that of thrombolysis in the acute phase (Vaitkus, 1995; DARE-988078, 1999; "Primary angioplasty versus intravenous...", 2002; Weaver, et al., 1997; DARE-988115, 1999) [B] and also in long-term follow-up. Stenting probably improves the outcome (Meads, et al, 2000; DARE-20018012, 2002; Grines, et al., 1999) [A]. Further treatment with clopidogrel for 3 months.
- An ACE inhibitor to all patients with signs or symptoms of heart failure or ejection fraction (EF) [A]. Therapy is not usually started on the first day.
    - e.g., captopril. Start with 6.25 mg and increase the dose rapidly.
- Continuous nitrate therapy (Mehta & Yusuf, 2000) [A]
    - Administered as an infusion, if the patient has ischaemic pain and pain medication has no effect. Nitrate infusion.
    - Orally, e.g., isosorbide dinitrate 10 to 20 mg x 2 to 3
- Heparinization is often indicated, if the patient
    - needs prolonged bed rest and is clearly obese (thrombosis prophylaxis)
    - has atrial fibrillation (also permanent warfarin therapy)
    - has ventricular aneurysm (also permanent warfarin therapy)
    - has unstable angina pectoris
    - has embolic complications
- Anticoagulation with warfarin is often started in massive anterior infarction and when transient ischemic attack (TIA) or stroke (mural thrombosis) occurs with MI.

Arrhythmias in Myocardial Infarction

Objectives

- To prevent sudden death and treat severe arrhythmias immediately
- To prevent arrhythmias by treating the underlying conditions

Causes of Arrhythmias

- Myocardial damage, ischaemia, and sympathetic stimulation are associated with ventricular arrhythmias.

- Ejection failure causes supraventricular tachyarrhythmias and atrial fibrillation.
- Vagal stimulation causes bradyarrhythmias and atrioventricular (AV) conduction disturbances, especially in cases of inferior-posterior wall infarction.
- Reperfusion often causes benign ventricular rhythm but also severe ventricular arrhythmias.

Ventricular Fibrillation

- Often occurs within 2 to 4 hours of infarction. After 12 hours, a primary ventricular fibrillation is rare.
- An early ectopic beat may initiate ventricular fibrillation in an ischaemic myocardium. Ectopic beats are not treated if cardiac monitoring is effective.
- Treatment
    - Acute ventricular fibrillation is treated by immediate defibrillation starting with 200 joules. Prolonged ventricular fibrillation frequently calls for cardiopulmonary resuscitation (CPR).
    - To prevent recurrence of fibrillation, lidocaine is given: initially as bolus of 100 mg, which can be repeated if necessary. Thereafter, a continuous infusion of 3 to 4 mg/min is given. Amiodarone is a modern and more effective alternative to lidocaine: infuse a 150 to 300 mg bolus in 20 minutes. Thereafter infusion at 800 to 1200 mg/24 hours.
    - A beta-blocker is usually added to the therapy.

Ventricular Tachycardia

- More than three ectopic beats and a heart rate over 120 beats/min.
- Brief, spontaneously ending spurts are seen in over 50% of patients with infarction during the first two days. They occur mainly after 8 to 14 hours, not immediately after the infarction, as ventricular fibrillation does.
- Ventricular tachycardia leads to haemodynamic collapse or ventricular fibrillation. The severity depends on the duration, variability, frequency, and timing of tachycardia.
- Ventricular tachycardia may be monomorphic or polymorphic
- Treatment
    - Beta-blocker
    - Lidocaine boluses and infusion as in ventricular fibrillation, if haemodynamics is compromised. Amiodarone may be a better alternative.
    - If necessary, synchronized cardioversion shock with 50 joules is performed.
    - Late in infarction, ventricular tachycardia is, like ventricular fibrillation, a serious problem that requires further examination.

Ventricular Ectopic Beats

- Occur in nearly all patients with painful MI
- May cause complications if they are frequent (more than 5/min), are variable or occur concomitantly with an early T wave
- Treatment is usually not necessary if cardiac monitoring is effective. A beta-blocker may be indicated. Potassium level should be kept above 4.0.

Idioventricular Rhythm

Idioventricular rhythm is an arrhythmia often associated with MI. In the reperfusion phase, it may even indicate that thrombolysis has been successful. The frequency is often 70 to 80 bpm and drug therapy is not necessary.

Supraventricular Tachyarrhythmias

- Atrial fibrillation in a patient with infarction is often associated with cardiac insufficiency and worsens the prognosis. Atrial fibrillation increases the risk of stroke, which is why low molecular weight (LMW) heparin and warfarin therapy are indicated.
- Atrial fibrillation is often associated with the thrombosis of the right coronary artery or the circumflex branch: reperfusion also often corrects atrial fibrillation.
- Atrial function is important in MI. In cardiac insufficiency, rapid atrial fibrillation requires active direct current (DC) cardioversion. Often, the achieved sinus rhythm does not last. In such a case, haemodynamics must be stabilised (oxygenation, treatment of pulmonary oedema, controlling of ventricular response with a beta-blocker and digitalis) after which spontaneous reversal of the rhythm is waited for. The effect of the beta-blocker is seen rapidly but that of digitalis not before several hours. Rapid ventricular response may be controlled even if cardiac insufficiency is present: the benefit often outweighs the disadvantage.
- Selective beta-blockers are best suited for maintaining the achieved sinus rhythm.
- Intravenous amiodarone will not reduce the contraction of the myocardium. It is effective in prophylaxis of atrial fibrillation (together with a beta-blocker) and it may be used in cardioversion of atrial fibrillation and/or slowing down the ventricular response.
- Ibutilide is a new class III drug with a single indication: treatment of atrial fibrillation and flutter. There are limited data on its use in patients with infarction.
- Note: A broad QRS complex tachycardia in a patient with infarction must always be treated as a ventricular tachycardia.

Bradyarrhythmias

- A strong vagal reaction in the early stages of infarction may lead to a circulatory collapse.
- Postero-inferior wall infarction is often associated with a functional atrioventricular block. The QRS complex is narrow and the heart rhythm is 50 to 60 even in cases of a total block. A pacemaker is rarely needed.
- In anterior wall infarction, the proximal conduction system may be blocked: the QRS complex is wide, the substituting rhythm is slow (30-40), the patient is in a poor condition and pacing is necessary. Prognosis is poor even with pacing.
- Drug treatment
    - atropine 0.5 mg i.v., repeated as necessary, for treatment of functional bradycardia.

Pacemaker

- In anterior wall infarction, pacing is indicated if there is a 2nd or 3rd degree block. Pacing should be anticipated in case of a trifascicular block, alternating right and left bundle branch block, or if an extensive infarction is associated with left anterior hemi-block (LAFB) or left posterior hemi-block (LPFB).
- Postero-inferior wall infarction associated with a 3rd degree atrioventricular block requires pacing if bradycardia is detrimental to haemodynamics and not responsive to treatment with atropine.
- Sinus bradycardia may be temporarily controlled with i.v. atropine.

Circulatory Conditions and Their Treatment after Myocardial Infarction

Condition and treatment	Symptoms and signs
Normal circulation • Monitoring • i.v. line (saline drop)	• heart rate and blood pressure normal • no arrhythmias • no heart insufficiency
Hyperdynamic state • beta-blocker (metoprolol, atenolol, practolol 2 to 5 mg i.v.)	• increased heart rate, high blood pressure
Neurovascular reflex (bradycardia-hypotension) • atropine 0.5 mg i.v., repeated ad 2 mg • dopamine infusion, if necessary	• usually in connection with postero-inferior infarction • bradycardia, hypotension
Hypovolemia • 0.9% saline 200 ml in 5 to 10 minutes according to the response	• low blood pressure, low central venous pressure (CVP), tachycardia • cold extremities • decreased venous distension (also jugular veins)
Severe heart failure • nitrate infusion • dopamine infusion • continuous positive airway pressure (CPAP) • treatment of pulmonary oedema	• low blood pressure • cold extremities • engorged neck veins • chest crackles • chest radiograph

Treatment in Hospital

Follow-up and Treatment

- Pain: morphine, nitro, beta-blocker
- Blood pressure
- Skin, peripheral circulation
- Increased respiratory rate suggests cardiac insufficiency.
- Monitoring of arrhythmias
- ST segment changes
- Oxygen saturation; oxygen or continuous positive airway pressure
- A comfortable posture
- Informing and reassuring the patient
- Nicotine replacement therapy is started already in the hospital. Nicotine addiction may be evaluated by using the Fagerstrom test, and the planning of further treatment may be based on it.
- In an uncomplicated infarction, patients are allowed to sit as soon as they want, they can eat unassisted, and they can be helped to a portable toilet at the bedside. Intensive monitoring is usually needed for 1 to 2 days.
- The infarction is complicated and treatment lasts longer if the patient has had
    - shock
    - hypotension
    - obvious cardiac insufficiency (usually requires thrombosis prophylaxis or anticoagulation, especially if in connection with atrial fibrillation)
    - prolonged chest pain
    - serious ventricular arrhythmias
    - thromboembolic complications
    - anatomical complications (papillary muscle dysfunction or rupture)
    - pericarditis on days 2 to 4.
- Treatment of the patient in primary health care (in a primary health care hospital) is justifiable if the patient's prognosis is otherwise poor: those who are permanent inpatients or otherwise severely disabled and for whom invasive treatment has not been planned.

Assessment of Risk Factors in a Patient with Myocardial Infarction

- The most important causes of mortality are
    - reinfarction
    - cardiac insufficiency
    - arrhythmias
- During hospitalization, a poor prognosis is indicated by
    - cardiac insufficiency and extensive infarction (ejection fraction [EF] <25%)
    - chest pain and ischaemic ST changes (send to angiography)
    - in connection with non-Q-wave infarction, risk factors for coronary heart disease (CHD) and especially diabetes mellitus
- Evaluation of ischaemia and need for active treatment
    - Risk is highest during the first few weeks and months after infarction. Therefore, at the end of the hospital treatment, an early symptom-limited exercise test is performed on many patients to estimate the need for angioplasty and coronary surgery in particular.
- For indications of coronary angiography, see the NGC summary of the Finnish Medical Society Duodecim guideline Coronary Angiography and Indications for CABG or Angioplasty.

Care after Myocardial Infarction

Drug Treatment

- ASA, beta-blocker (Freemantle, et al., 1999; DARE-999336, 2001; Sudlow et al., 2002) [A], ACE inhibitors, and statins have been shown to improve the prognosis. Glycaemic control is also important.
- Unnecessary drugs instituted during the initial phase should be discontinued already towards the end of hospital treatment or when the patient comes to the first check-up, not on the last day in hospital.
- Only those with cardiac insufficiency or poorly controlled blood pressure need a diuretic.
- ASA 50 to 100 (-250) mg is given unless there are contraindications ("Collaborative overview of randomised trials of antiplatelet therapy", 1994; DARE-948032, 1999) [A].
- Patients with hypertension, angina pectoris, ventricular arrhythmias, ischaemia during an exercise test, previous infarction, an enlarged heart, low ejection fraction, or a cardiac insufficiency need a beta-blocker. In practice, these drugs are given to all patients who have no contraindications. Adequate beta-blockade is achieved when the heart rate at rest is about 60 bpm.
- Nitrate plus a beta-blocker are given to all patients with angina pectoris or ischaemia during an exercise test. Nitrate is a drug used for symptom relief that can often be discontinued.
- An ACE inhibitor is given to all patients with clear systolic dysfunction (ejection fraction less than 40%) (Sudlow et al., 2002) [A]. A milder systolic dysfunction is treated with an ACE inhibitor if the patient has cardiac insufficiency (symptomatic or asymptomatic), valvular regurgitation, hypertension, or diabetic nephropathy. The indications of ACE inhibitors have been constantly extended, and they are now given to almost every patient who has had an infarction. So-called "asymptomatic cardiac insufficiency" and even secondary prevention (according to the Heart Outcomes Prevention Evaluation [HOPE] study) in high-risk patients have become indications (Yusuf et al., 2000). ACE inhibitor therapy may be more difficult if the patient has a valvular obstruction, hypotension, or uraemia. Patients on diuretics have a risk of hypotension, especially when treatment with an ACE inhibitor is started. The ACE inhibitor dose should not remain at the level of the initial dose unless hypotension and creatinine elevation prevent the titration.
- A lipid-lowering drug (a statin) is given to all patients with serum low-density lipoprotein (LDL) cholesterol >3.0 mmol in spite of the diet (Rembold, 1996; DARE-961089, 1999) [A]. For calculation of the level, see the LDL Cholesterol calculator program available on the EBM CD-ROM and the EBM Web site.
- An anticoagulant is given if the patient has atrial fibrillation, an embolic complication, or ventricular aneurysm verified by echocardiography, often also short-term in the treatment of an extensive anterior wall infarction.
- Elevated serum homocysteine concentration is associated with cardiovascular diseases, but it does not appear to predict arterial disease in healthy persons (Knekt et al., 2001) [C]. However, homocysteine concentration correlates well with blood pressure, cholesterol concentration and smoking, and is thus a good indicator of the intensity of the atherosclerotic process. Folic acid (both vitamin B6 and B12) lowers serum homocysteine concentration, but evidence of its slowing effect on the progression of atherosclerosis is scarce (only one study in which the use of vitamins after percutaneous transluminal coronary

angioplasty reduced the occurrence of re-stenosis) (Schnyder, 2002) [B]. Several studies are currently ongoing on secondary prevention.
- A quiet moment should be reserved for discussing life after MI and living with coronary artery disease (CAD) while the patient is still in the hospital.
    - Such a discussion helps to reduce psychological problems and disability.
    - Give instructions for dealing with possible exacerbation of the disease.
    - The motivation to quit smoking is highest after an infarction:
        - nicotine replacement therapy according to individual evaluation (Fagerstrom test)
    - A cholesterol and saturated fatty acid-restriction diet and/or drug treatment
    - Exercise counseling according to individual evaluation: the patient must be able to talk while exercising.
    - Rehabilitation course
    - Secondary prevention

Sick Leave

- Duration 2 to 3 months
- Re-examination after about one month, usually within specialist health care.
    - History of symptoms: if the patient has had angina pectoris symptoms, consider testing exercise capacity, if the test has not been performed yet.
    - Remind the patient of the principles of healthy life style.
    - Serum lipids should be measured if they were high on an earlier measurement.
    - Control the adequacy of beta-blockade: target heart rate 50 to 60 bpm.
    - Possible depression should be diagnosed.
- The ability to work is evaluated before the end of the sick leave. If necessary, an exercise test is carried out to assess working ability.

Related Evidence

- Glucose-insulin-potassium probably reduces mortality in acute MI. However, its role in combination with thrombolysis or acute revascularization should be determined by larger randomized trials (Fath-Ordoubadi & Beatt, 1997; DARE-971070, 1999) [B].
- There is little evidence from randomized trials of any significant further net clinical benefit from adding either subcutaneous or intravenous unfractionated heparin to the treatment of patients who are given aspirin (Collins et al., 1996; DARE-978036, 1999) [B].
- Low-dose amiodarone may have a beneficial effect on total mortality after MI, but the drug has many adverse effects (Zarembski et al., 1993; DARE-940032 1999) [C].
- Class I antiarrhythmic agents increase the risk of death after MI (Sudlow et al., 2002) [A].
- Sotalol increases mortality in patients with MI who have left ventricle failure (Sudlow et al., 2002) [B].

- The evidence does not support the hypothesis that verapamil use is associated with harm in patients with MI (Pepine, Faich, & Makuch, 1998; DARE-981601, 2000) [B].
- Exertion-related MIs occur in habitually inactive people with multiple cardiac risk factors (Giri et al., 1999) [B].

Definitions:

Levels of Evidence

A: Strong research-based evidence. Multiple relevant, high-quality scientific studies with homogeneous results.

B: Moderate research-based evidence. At least one relevant, high-quality study or multiple adequate studies.

C: Limited research-based evidence. At least one adequate scientific study.

D: No research-based evidence. Expert panel evaluation of other information.

CLINICAL ALGORITHM(S)

None provided

EVIDENCE SUPPORTING THE RECOMMENDATIONS

REFERENCES SUPPORTING THE RECOMMENDATIONS

References open in a new window

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

Concise summaries of scientific evidence attached to the individual guidelines are the unique feature of the Evidence-Based Medicine Guidelines. The evidence summaries allow the clinician to judge how well-founded the treatment recommendations are. The type of supporting evidence is identified and graded for select recommendations (see the "Major Recommendations" field).

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

- Appropriate diagnosis and treatment of myocardial infarction and associated arrhythmias
- Reduction in mortality and morbidity
- Improved long-term prognosis

Subgroups of Patients Within Target Population Most Likely to Benefit from These Recommendations

- Beta-blockers are useful especially in patients who are tachycardic and hypertensive but do not have heart failure.
- The benefit of fibrinolytic therapy is highest among patients with bundle branch block or ST elevation. Patients with ST depression or other electrocardiographic abnormalities showed no conclusive evidence of benefit.

POTENTIAL HARMS

- Fibrinolytic therapy was associated with a small but significant excess of 3.9 (standard deviation 0.8) strokes per 1000 patients. All of this excess appeared on days 0 to 1. Fibrinolytic therapy was associated with a 7.3 (standard deviation 0.7) per 1000 excess of nonfatal major bleeds.
- Amiodarone. Reports of fatal complications of the drug use exist. 30% of patients in the trials reported side effects versus 10% in the control groups. Drop out rates were 8 to 20.8% due to side effects.

CONTRAINDICATIONS

CONTRAINDICATIONS

- Aspirin (acetylsalicylic acid). Contraindications include active ulcer, hypersensitivity to acetylsalicylic acid, and anticoagulation.
- Beta-blockers. Contraindications include asthma, hypotension, heart insufficiency, conduction disturbance, bradycardia.
- Angiotensin-converting enzyme inhibitors. Contraindications include hypotension and uraemia. Concomitant use of calcium channel blockers should be avoided because of risk of hypotension. Patients on diuretics have a risk of hypotension, especially when treatment with an angiotensin-converting enzyme inhibitor is started.

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better

IOM DOMAIN

Effectiveness
Patient-centeredness
Timeliness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

Finnish Medical Society Duodecim. Myocardial infarction. In: EBM Guidelines. Evidence-Based Medicine [CD-ROM]. Helsinki, Finland: Duodecim Medical Publications Ltd.; 2003 Jul 11 [Various]. [30 references]

ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

2001 Apr 30 (revised 2003 Jul 11)

GUIDELINE DEVELOPER(S)

Finnish Medical Society Duodecim - Professional Association

SOURCE(S) OF FUNDING

Finnish Medical Society Duodecim

GUIDELINE COMMITTEE

Editorial Team of EBM Guidelines

COMPOSITION OF GROUP THAT AUTHORED THE GUIDELINE

Primary Author: Editors

FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

Not stated

GUIDELINE STATUS

This is the current release of the guideline.

This guideline updates a previous version: Myocardial infarction. Helsinki, Finland: Duodecim Medical Publications Ltd; 2002 Mar 30. Various p.

GUIDELINE AVAILABILITY

This guideline is included in a CD-ROM titled "EBM Guidelines. Evidence-Based Medicine" available from Duodecim Medical Publications, Ltd, PO Box 713, 00101 Helsinki, Finland; e-mail: info@ebm-guidelines.com; Web site: www.ebm-guidelines.com.

AVAILABILITY OF COMPANION DOCUMENTS

- EBM guidelines. Evidence-based medicine. Helsinki, Finland: Duodecim Medical Publications, Ltd. 2002. [CD-ROM]
- EBM guidelines. Web site: www.ebm-guidelines.com.

Available from: Duodecim Medical Publications, Ltd, PO Box 713, 00101 Helsinki, Finland; e-mail: info@ebm-guidelines.com; Web site: www.ebm-guidelines.com.

PATIENT RESOURCES

None available

NGC STATUS

This summary was completed by ECRI on August 28, 2001. The information was verified by the guideline developer as of October 26, 2001. This summary was updated by ECRI on December 9, 2002. This summary was verified by the developer on April 2, 2003. This summary was updated again by ECRI on December 29, 2004.

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